SUPPLEMENT TO
THE MEDICAL JOURNAL OF AUSTRALIA
Number 22, August 21, 1943.

WAR MEDICINE AND SURGERY

Compiled under the direction of the Committee on the Survey of War Medicine of the National Health and Medical Research Council.

BACILLARY DYSENTERY.

It has been claimed that the main factor which prevented the Allies from taking Gallipoli during the Great War was the high incidence of bacillary dysentery amongst their troops.[1] Even in that campaign, however, when the organism concerned was chiefly the more dangerous Shiga type, the death rate from the disease was not higher than 10%. In other circumstances, when facilities for treatment have been better and the Flexner organisms have predominated, the mortality has been quite low; for example, during the present war there has been reported a series of almost 2,000 patients amongst whom there were only two deaths.[2] Bacillary dysentery has therefore not caused a serious loss of life among adults, and, with the advent of sulphonamides, is even less liable to do so in the future. It is nevertheless an important cause of morbidity amongst troops, especially when the conditions of a campaign make it difficult to maintain the usual standard of hygiene; and once a man has contracted the disease, treatment in hospital is required for a period averaging three to four weeks.

It is worth while reviewing the present knowledge of the whole subject of bacillary dysentery, but stress will be laid on the following three items which represent the most important advances reported during the last few years. (*a*) Sulphonamide drugs have proved successful in the treatment of both patients and carriers. At the same time as this treatment has come to the fore, there has been a tendency to abandon the traditional methods of purgation. This is partly the result of the stress of competition from the sulphonamides, for the administration of saline purges to patients with dysentery can now be examined with a critical detachment which was hardly possible when it was the only method of treating the system directly affected. It is also perhaps partly due to the fact that there has been an increased dissemination of knowledge about the abnormal physiology of dehydration; the dangers of excessive loss of fluid are now well known, and are believed to outweigh the advantages of sweeping toxins from the small bowel, even though this latter procedure has a direct appeal which anybody can understand. (*b*) Although the MacConkey plate has in the past proved very valuable for the isolation of pathogenic organisms from bowel contents, it is now regarded as unsatisfactory for the culture of dysentery bacilli.[3] Wilson and Blair[17] on the one hand, and Leifson on the other,[3] have introduced differential media which have been modified by various workers,[3][7] but which represent an important advance in the methods of growing the dysentery bacillus. With the help of these media it has been possible to obtain more accurate information about carriers of dysentery bacilli. (*c*) In 1940 Boyd introduced a classification of the dysentery bacilli which is of very great interest and which forms a useful background to the understanding of the disease.[1]

Bacteriology of the Dysentery Bacilli.

Types of Dysentery Bacilli.

The following organisms are now accepted as causing dysentery: *Bacterium dysenteriæ* Shiga, *Bacterium dysenteriæ* Schmitz, *Bacterium dysenteriæ* Flexner and *Bacterium dysenteriæ* Sonne. They are non-motile, non-sporing, non-capsulated, Gram-negative rods which do not ferment lactose. They can be classified on the basis of their biochemical reactions as follows, though there is an element of untrustworthiness about most fermentation reactions.

1. Non-mannitol-fermenting bacilli. These include the groups *Bacterium dysenteriæ* Shiga and *Bacterium dysenteriæ* Schmitz, of which the latter alone produces indole in a broth culture. The Shiga bacillus is associated with the most severe form of bacillary dysentery, owing to the exceedingly potent toxin it produces. It has not so far become endemic in Australia, but it has been isolated here on a few occasions, and its further introduction in the near future has been described as "inevitable".[14] Some of our soldiers suffered from it in the Middle East and others no doubt are in danger of contracting it from the Japanese, amongst whom it is a relatively common cause of dysentery. The Shiga and Schmitz organisms have a distinct and uniform antigenic structure, so that strains isolated in one part of the world are agglutinated by serum prepared elsewhere. There is no such uniformity amongst the mannitol-fermenting organisms.

Certain strains, which resemble *Bacterium dysenteriæ* Shiga and *Bacterium dysenteriæ* Schmitz in their biochemical reactions, but which do not become agglutinated with antiserum prepared against these organisms, have recently been reported as occurring in patients with bacillary dysentery in India and Egypt.[13] They have been divided into eight distinct types.

2. Mannitol-fermenting bacilli which do not ferment lactose or saccharose. These organisms form a composite group which can be subdivided in part by fermentation reactions, but which require serological examination for their full classification. The serological types described in the past have been five: Flexner V, Flexner W, Flexner X, Flexner Y and Flexner Z. Boyd has pointed out that, in this subdivision, a distinction is not made between the group antigen and the various type antigens. He has found it possible to establish six types, each of which has its specific type antigen as well as a common group antigen. These types are named Flexner I, Flexner II, Flexner III, Flexner IV, Flexner V and Flexner VI.

Boyd has also described other organisms which do not possess the group antigen referred to above and have not a common group antigen of their own, but which show the typical Flexner biochemical reactions and have specific type antigens. It has been decided that the organisms which have these characteristics and which are also true dysentery bacilli, should be called the Boyd group; they have been subdivided into Boyd I, Boyd II and Boyd III types, and there are at least three others which up to 1940 Boyd had not shown capable of causing dysentery. Last year an outbreak of dysentery in Queensland was found to be due chiefly to a mannitol-fermenting organism which was one of the three investigated but not incriminated by Boyd. In the light of this further experience the organism has been included in the pathogenic group and has been tentatively named Boyd IV.[12] The same organism has meanwhile proved quite common among soldiers with dysentery in the Middle East, and investigations are being carried out to determine the extent of its occurrence in Australia and New Guinea.[13]

Several small outbreaks of dysentery in England have been caused by an organism which has been named the Newcastle bacillus after the city in which it was first isolated. This strain has a general resemblance to the Flexner organisms, but has three important individual characteristics: it produces gas in the sugars it ferments, especially in glucose; it is occasionally motile; and it does not produce indole. It has been included as one of the six Flexner types.

3. Mannitol-fermenting bacilli which are also late fermenters of lactose and saccharose. This group includes the Sonne organisms which cause a mild type of dysentery. The fact that these bacteria ferment lactose, even though they take about six days to do so, is important, since such fermentation is usually regarded as a sign that a coliform organism is non-pathogenic. The Sonne and Newcastle bacilli each have some characteristics which suggest that they are intermediate between the saprophytic *Bacterium coli* and the highly pathogenic Flexner and Shiga strains.

4. Other organisms. In addition to the accepted members of the dysentery group there are a number of other organisms which are sometimes isolated from dysenteric stools and which may be regarded as occasionally pathogenic. They include *Bacillus Morgani*, *Bacillus alkalescens*, *Bacillus pyocyaneus* and *Bacillus dispar*. It is said that in young children *Bacillus coli* may sometimes cause inflammatory diarrhœa.[32]

Isolation of Dysentery Bacilli from Fæces.

The organisms can be isolated most easily in the first twenty-four to forty-eight hours after the onset of symptoms. Later in the disease secondary invaders become prominent and the dysentery bacillus becomes relatively scarce. Nowadays this is not the important disadvantage that it used to be, for the new selective media are able to inhibit most of the non-dysentery organisms.

After a stool has been passed the dysentery bacilli contained in it die out very quickly unless care is taken to preserve them. The simplest method is to collect the stool at once and make the cultures within half an hour, but if for any reason delay is unavoidable, some method of preservation of the sample should be attempted. If dehydration can be prevented and a slightly alkaline environment maintained, the dysentery organisms, especially Flexner and Sonne groups, will probably remain alive at 15° C. for several days. A suitable preservative can be made with the following: 300 cubic centimetres of glycerin; 700 cubic centimetres of distilled water; 6 grammes of sodium chloride; 3·1 grammes of dipotassium hydrogen phosphate; and 1 gramme of potassium dihydrogen phosphate. The mixture is tubed and autoclaved and the final solution should be sufficiently alkaline to give a pink colour with phenol red (pH 7·4).

It is essential to select a portion of mucus from the stool and inoculate the plate with this mucus rather than with fæces. The latter apparently contain substances which inhibit the dysentery bacilli, so that they are not the optimal material for culture even if the excess of colon organisms can be controlled by the use of selective media. The piece of mucus is fished out from the stool by means of a wire loop and is washed in two portions of sterile saline solution to remove adherent fæces. It is then seeded onto an appropriate medium, the surface of which must be dried before the inoculation.

The desoxycholate-citrate medium introduced by Leifson[9] and modified by others[5][17] is much better than MacConkey's medium for the demonstration of dysentery organisms in fæces, and there is no justification for the continued use of MacConkey's agar, so far as work on the dysentery bacillus is concerned.[9] The Leifson medium suppresses almost completely most coliform organisms, and to a less extent paracolon and atypical coliform organisms; it has little action on Proteus strains. It favours the growth of all types of dysentery bacilli, including the Sonne group, for which Leifson himself had thought the media unsuitable: he had apparently used old, rough Sonne strains as test organisms, whereas the strains usually encountered in the examination of fæces are smooth. The preparation of the medium must be carefully controlled.

Another selective medium contains lactose agar to which has been added potassium tellurite, iron alum and rosolic acid.[27] It allows *Bacterium dysenteriæ* Flexner to develop and inhibits most coliform strains. It also permits the growth of the other dysentery bacilli, though the Sonne group develops slowly. The selective media have the advantage of being easy to work with as well as very reliable. Their relative superiority is increased when the dysentery organisms are reduced in numbers, as, for example, in carriers and in patients with chronic dysentery.

After twenty-four hours' incubation the plates are examined and suspicious colonies are subcultured onto agar, broth, milk and a series of carbohydrate media such as lactose, glucose, mannitol and dulcite. The growth is frequently so luxuriant, however, that colonies can be picked off and direct slide-agglutination with the appropriate antiserum can be carried out at once. In this case the bacteriologist can make or confirm the diagnosis within twenty-four hours of receiving the specimen. On the following day confirmation is obtained by tentative readings of the fermentation reactions and by a test for motility.

Clinical Course.

It has already been stated that Shiga organisms often produce a severe infection, Sonne organisms a mild diarrhœa, and Flexner bacilli an illness of intermediate severity. These are generalizations, however, and are not necessarily true of any particular case.

The disease may be arbitrarily classified into mild, moderate and severe types.

The severe form is so overwhelming that the patient may die in about forty-eight hours from the commencement. The stools contain dark clotted blood and gangrenous sloughs, but mucus is absent because of destruction of the goblet cells. Ominous signs are: collapse with subnormal temperature; or sustained high fever of intermittent or remittent type; or the development of hiccup. There are other types, however, in which a completely unexpected fatal ending occurs; for example, the patient may have a mild afebrile illness for seven to fourteen days and then suddenly become very ill and die; or, on the other hand, a fever of a couple of weeks' duration may disappear, but the patient may gradually grow worse and die. The severe type of dysentery was not common in the recent Middle East campaigns where, judging by several published reports, the over-all death rate must have been rather less than 1%.

In the moderately severe type, the onset is usually sudden. The patient feels a colicky pain in the abdomen, he may vomit, and soon afterwards diarrhœa begins. The temperature is raised, the pulse rapid, the tongue furred, and there are often pains in the limbs and frontal headache. The stools soon lose their fæculent character and become first, watery evacuations with shreds of thin mucus, and then, very small motions consisting almost entirely of sticky mucus, like raw white of egg, flecked with blood. They may number five to fifty a day, so that the patient rapidly becomes dehydrated, as is shown by thirst, sunken eyes, loss of weight, dry, brown tongue, low venous pressure, restlessness, oliguria and nitrogen retention. There is often a generalized abdominal tenderness, but in some patients tenderness is marked only over the sigmoid colon. Tenesmus is common and micturition difficult and painful. The anus may become inflamed, excoriated and sore, especially since drinking, eating and every movement intensify the desire to defæcate. As a rule the general symptoms subside in 48 hours, and over a period of ten to fourteen days the stools become fewer and more fæculent and lose their blood and mucus.

The mild type of infection is characterized by diarrhœa without much constitutional disturbance. The temperature rises slightly, but soon returns to normal. The stools consist of liquid fæces with mucus and occasional streaks of blood. Improvement occurs within twenty-four hours. The complaint is often considered to be due to unaccustomed food or climate, and is given names such as "Gyppy tummy" which vary with the country in which it occurs.

Complications and Sequelæ.

The disease sometimes becomes chronic: diarrhœa and the passage of blood and mucus persist, while emaciation and anæmia become progressively greater. In other patients there is a tendency to recurrence of diarrhœa, but melena is absent, though copious mucus may be passed. This condition is often neurotic in origin and results from fear engendered by the original sickness.

Arthritis occurs in as many as 25% of patients in some epidemics, but is quite rare in others. It was common amongst German troops in Poland in 1939. The symptoms generally do not develop until the diarrhœa has ceased for at least a few days, and their severity is not necessarily parallel with that of the dysentery. The attack is often associated with pyrexia and rigors and may involve one joint or many, the knee being the joint most frequently affected. There is a large effusion and considerable muscular atrophy, but suppuration does not occur, and pain is not severe. Complete recovery generally results, but not until several months have passed. The condition is usually considered to be allergic in nature, though occasionally there have been reports of the recovery of dysentery bacilli from the synovial fluid of the affected joint.

Other complications include peripheral circulatory failure; conjunctivitis, which occurs about the tenth day; iritis, which usually begins about the second week; true glomerulo-nephritis; toxic nephrosis; peripheral neuritis; perforation of a dysenteric ulcer; intestinal hæmorrhage; metastatic infections such as bronchopneumonia, pyelitis, parotitis, or portal pyæmia; hæmorrhoids; and rectal prolapse.

Diagnosis.

A diagnosis of bacillary dysentery may be made on clinical grounds when the disease has had a sudden onset and has been characterized by fever, abdominal pain and frequent stools containing blood and mucus. This diagnosis must be supported whenever possible by bacteriological findings and a microscopic examination of the fæcal exudate. The former type of evidence may, of course, be quite conclusive, but the latter provides fairly reliable information without any delay, and this is an obvious advantage in the diagnosis of a disease that has a rapid course.

A piece of mucus is said to come from a "typical bacillary exudate" when 90% of the cells, excluding the erythrocytes, are polymorphonuclear cells. The remaining 10% consist mainly of epithelial cells and large macrophages, which may contain red blood cells and look very much like *Entamœba histolytica*, but which are, however, non-motile. This diagnostic type of exudate is usually seen only in the first few days of the disease. Later on the polymorphonuclear cells autolyse and become relatively fewer; then the exudate has to be classified as "indefinite" and does not help in diagnosis. Sometimes there may not be an exudate.

The details of the isolation of the dysentery bacilli have already been given. The determination of the type of Flexner organism is usually unnecessary unless epidemiological information is being sought.

A sigmoidoscopic examination may give valuable data in chronic dysentery, but should never be undertaken when the disease is in the acute stage. The healthy mucosa appears pink and glistening, but in chronic dysentery the colour is a deeper red than normal, and the surface is covered with a mucoid exudate. One may see shallow ulcers of various sizes and shapes with the bases covered by a layer of pus and the edges undermined.

Examination of the patient's serum for antibodies against the various types of dysentery bacilli may be made. The multiplicity of antigenic varieties complicates the test and it is used mainly for chronic dysentery, in which condition there is less likelihood of isolating the organism. In any case, the agglutinin titre is not significantly high until about the tenth day. As in similar investigations on other organisms, the most conclusive result is the finding of a rising titre on repeated examination.

Differential Diagnosis.

There are many conditions which are liable to simulate bacillary dysentery and which have to be distinguished from it. (*a*) Amœbic dysentery is usually an afebrile illness of gradual onset and chronic course. The stools are rarely mucoid, but contain fæcal matter, and are therefore more offensive in smell. Examination of the stool reveals *Entamœba histolytica*. (*b*) Bilharzial dysentery has a definite geographical range, and the causative organism, *Schistosoma mansoni*, produces a characteristic ovum with a lateral spine. (*c*) Malarial dysentery may be due to a subtertian infection, in which the intestinal capillaries have been blocked by malarial parasites. It sometimes happens, however, that dysentery bacilli attack a person whose resistance has been lowered by malaria and the two diseases occur concurrently. The finding of malarial parasites, therefore, does not necessarily give the explanation of the dysentery. (*d*) Paratyphoid infection may cause ulceration of the large intestine with blood and mucus in the stools, and may be diagnosed as dysentery until the paratyphoid organisms are discovered. (*e*) Food poisoning should be suspected if vomiting is the initial symptom and the illness occurs simultaneously in a number of people who have eaten from the same food supply. (*f*) *Polyposis coli* sometimes resembles dysentery, but can frequently be diagnosed by sigmoidoscopy. (*g*) Carcinoma of the sigmoid flexure or rectum may be detected by radiological examination, in the former case, and digital or sigmoidoscopic examination in the latter. (*h*) Ulcerative colitis is indistinguishable clinically from chronic bacillary dysentery and may, in fact, be the same condition. (*i*) Mucous colitis has already been described as a functional disorder which sometimes follows an attack of bacillary dysentery. (*j*) There are certain rather rare causes of dysentery, such as mercury poisoning, arsenical poisoning, tuberculosis of the colon, uræmic colitis, giardiasis and diverticulitis.

Prophylaxis.

Bacillary dysentery has a world-wide dissemination. It is found wherever two essential conditions are present—carriers and poor sanitation. Frequently it has been associated with tropical and subtropical climates; and it is true that, in so far as these climates are more suitable than temperate zones for the breeding of flies, they provide greater opportunities for transmission of the bacilli to human food. But flies are not obligatory for the spread of bacillary dysentery; an outbreak of the disease occurred, for example, amongst the members of Byrd's polar expedition. From the point of view of prophylaxis, therefore, attention may be concentrated on dysentery carriers, methods of sanitation, and the number of susceptible subjects.

Dysentery carriers are more common than used to be thought a few years ago, when the methods of detecting them were inadequate. In a recent investigation of 26 patients convalescing from dysentery, platings on MacConkey agar seemed to indicate that all were free of organisms by the fortieth day, whereas the desoxycholate-citrate agar showed that five patients were still infective at that time.[9] The full results of this investigation indicate that convalescents continue to excrete *Bacterium dysenteriæ* Sonne for considerably longer periods than we have been led to believe in the past, and consequently patients have been discharged from hospitals earlier than they should have been. It has been found that more than half the clinically infected patients still excrete the organism three to four weeks after the onset of the disease. Some workers now require eight consecutive "negative" stools in four weeks before they discharge either a patient or a symptomless carrier from isolation, though three consecutive failures to produce a culture have been accepted in the past.

By means of the improved media it has also been shown that symptomless carriers often exceed in numbers the currently occurring cases. This is a well-known conception so far as diseases such as infantile paralysis are concerned, but it is new in its application to bacillary dysentery.

The discovery of carriers, of course, should be followed by the treatment of them. It has been shown that sulpha-

guanidine rapidly terminates the carrier state in Flexner infections,[10] though it is not so effective against the Sonne organisms.

The detection of carriers is difficult enough in civilian life, but is a much more formidable undertaking amongst army personnel; for in the army the laboratory facilities sometimes do not permit bacteriological examinations to be made even for diagnostic purposes.[13] In the army, therefore, great stress has to be placed on good hygiene as a means of nullifying the potential damage done by the undetected carrier.

Attempts have been made to render people immune to dysentery by preventive inoculation, but these measures have not so far been very successful. It has recently been reported that sulphaguanidine, given prophylactically in a dose of 0·5 gramme three times a day, abruptly stopped a spreading epidemic.[11]

Treatment of Acute Bacillary Dysentery.

The treatment of acute bacillary dysentery can be discussed under the following headings.[3]

1. *Nursing.*—Special wards should be set aside for these patients so as to prevent cross infection and to ensure the proper disposal of contaminated fæces. Rest in bed throughout the acute stage is essential; and dysentery pads made of cotton and tow or of gamgee tissue are more comfortable than bed pans during the period of intense diarrhœa. Care must be taken of the buttocks and back, to prevent soddenness and resulting infection. The stools must be examined at least once a day, for they provide the best guide to progress.

2. *Diet.*—Patients should receive nothing but water for the first twelve hours. They are then given small but frequent feeds of a non-residue, semifluid diet consisting of such foods as weak tea, biscuits, jelly, beef tea, chicken soup, "Bovril", "Ovaltine", arrowroot and sago. Milk is best avoided because the curds irritate the intestine. After a few days extra items such as eggs, bread, custard, stewed fruit and rice puddings may be added. When blood and mucus have disappeared from the fæces, other non-residue foods such as fish, chicken and minced steak can be included. Even when the patient is convalescent he should avoid pips and skins of fruit and all vegetables except *purées*. It is wise to put the patient on his ordinary diet for a few days before he is discharged from hospital.

3. *Drugs.*—Morphine is of great value in relieving pain and ensuring rest. Kaolin may be useful at a later stage.

4. *Saline Treatment.*—The use of magnesium sulphate or other saline purges has been strongly criticized during the last few years,[2][3][11] and it seems doubtful whether this orthodox treatment will persist much longer.

5. *Intravenous Infusions.*—It is logical in theory and successful in practice to relieve dehydration by the administration of fluid. Normal saline solution has proved effective for this purpose, but, since the patients lose protein in the blood and inflammatory exudate which enter the lumen of the bowel, there is an increasing tendency to give a transfusion of serum, plasma or whole blood.

6. *Specific Antiserum.*—The monovalent antiserum prepared against Shiga organisms is potent, but when one considers the large and increasing number of Flexner and Boyd organisms and the relative mildness of the disease they cause, it will be agreed that the use of a comprehensive polyvalent antiserum is not worth while. The dose of anti-Shiga serum is 50,000 to 100,000 units, and a modern concentrated product can be made to contain 20,000 units per cubic centimetre. The results of using this serum in the Middle East have been disappointing in that the benefit produced is only temporary.

7. *Sulphonamides.*—Two main queries have been made in regard to the use of sulphonamides in this disease: (*a*) whether they are really effective; and (*b*) whether sulphapyridine and sulphathiazole are not as useful as drugs such as sulphaguanidine and succinylsulphathiazole which have been specially developed as intestinal antiseptics.

The consensus of opinion is that chemotherapy is very effective against the dysentery bacilli; but a few competent clinicians have doubted this, and, since the disease is mild and self-limited in many cases, the sceptics have not been convinced that sulphonamides have decreased the mortality rate. The evidence in favour of these drugs, therefore, consists of at least five indications of clinical improvement following their use:[10] (*a*) within twenty-four to forty-eight hours of their administration there is an increased feeling of well-being, and relief of abdominal pain and tenesmus; (*b*) the temperature and pulse rate return to normal within a few days, in uncomplicated cases; (*c*) there is a remarkable reduction in the number of stools; (*d*) there is a rapid disappearance of blood from the fæces; and (*e*) the time spent in hospital is reduced.

Sulphapyridine and sulphathiazole have both been tried, and found successful, in the treatment of bacillary dysentery; but the special virtue of sulphaguanidine and succinylsulphathiazole is that they are effective in doses which produce very low blood concentrations and which are therefore less likely to cause general toxic actions than sulphapyridine or sulphathiazole. Sulphaguanidine and succinylsulphathiazole are equally effective; the former occasionally causes mild toxic symptoms such as headache, malaise and drug rash, whereas the latter has not been reported as having undesirable side actions; on the other hand, the greater availability of sulphaguanidine determines the issue between these two drugs at the present time. The dosage of sulphaguanidine recommended is: (*a*) an initial dose of 7·0 grammes; (*b*) a maintenance dose of 3·5 grammes every four hours until the fæces have been normal for a couple of days. If treatment is stopped too early the patients suffer a relapse.

References.

(1) J. S. K. Boyd: "Laboratory Diagnosis of Bacillary Dysentery", *Transactions of the Royal Society of Tropical Medicine and Hygiene*, Volume XXXIII, April, 1940, page 553.

(2) E. Bulmer: "Military Medicine in the Middle East", *British Medical Journal*, March 27, 1943, page 374.

(3) E. Bulmer and W. M. Priest: "Sulfaguanidine in the Treatment of Bacillary Dysentery", *Journal of the Royal Army Medical Corps*, Volume LXXIX, December, 1942, page 277.

(4) R. W. Fairbrother: "Selective Media for the Isolation of Bact. Dysenteriæ", *Journal of the Royal Army Medical Corps*, Volume LXXX, March, 1943, page 151.

(5) T. B. Gallie: "A Simple Medium for the Isolation of B. Dysenteriæ", *The Journal of Pathology and Bacteriology*, Volume LIV, April, 1942, page 256.

(6) B. C. Hobbs and V. D. Allison: "Report on an Institutional Outbreak of Sonne Dysentery and on the Value of Desoxycholate-Citrate Agar for the Isolation of Dysentery Organisms", *Bulletin of Hygiene*, Volume XVII, October, 1942, page 685.

(7) M. Hynes: "The Isolation of Intestinal Pathogens by Selective Media", *The Journal of Pathology and Bacteriology*, Volume LIV, April, 1942, page 193.

(8) E. Leifson: "New Culture Media Based on Sodium Desoxycholate for the Isolation of Intestinal Pathogens and for the Enumeration of Colon Bacilli in Milk and Water", *The Journal of Pathology and Bacteriology*, Volume XL, May, 1935, page 581.

(9) P. H. Manson-Bahr: "Dysentery and Diarrhœa in Wartime", *British Medical Journal*, September 19, 1942, page 346.

(10) L. Opper and V. Hale: "Sulfaguanidine in Treatment of Dysentery (Bacterium Flexneri) Carriers", *The Journal of the American Medical Association*, Volume CXIX, August 29, 1942, page 1489.

(11) J. W. Paulley: "Treatment of Bacillary Dysentery in the Middle East", *The Lancet*, November 21, 1942, page 592.

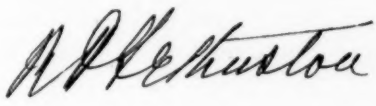
(12) L. E. Rothstadt, F. Fenner and B. A. Baker: "Bacillary Dysentery caused by Bacterium Dysenteriæ P274", THE MEDICAL JOURNAL OF AUSTRALIA, April 17, 1943, page 341.

(13) H. Sachs: "A Report on an Investigation into the Characteristics of New Types of Non-Mannitol-Fermenting Bacilli Isolated from Cases of Bacillary Dysentery in India and Egypt", *Journal of the Royal Army Medical Corps*, Volume LXXX, February, 1943, page 92.

(14) W. C. Sawers: "The Epidemiology of Bacillary Dysentery", THE MEDICAL JOURNAL OF AUSTRALIA, February 20, 1943, page 160.

(15) J. C. Scott: "Prophylactic Use of Sulfaguanidine", *The Journal of the American Medical Association*, Volume CXXII, June 26, 1943, page 588.

(16) Special Article: "Treatment of Bacillary Dysentery with Sulphaguanidine", *The Lancet*, January 3, 1942, page 20.

(17) W. J. Wilson and E. M. McV. Blair: "A Tellurite-Iron-Rosolic Acid Medium Selective for B. Dysenteriæ (Flexner)", *British Medical Journal*, October 11, 1941, page 501.

(18) E. H. Vere Hodge: "Modern Views of the Dysenteries and their Treatment", *The Practitioner*, Volume CXLVI, June, 1941, page 365.